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*"Scire est nescire, nisi id me
Scire alius sciret."*

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ANIMAL DISEASE IN RELATION TO MAN*

BY

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Man is so much in contact with animals that it is not surprising that at times he contracts disease from them. Much work remains to be done on diseases in which the part an animal plays is still unknown. It is impossible to cover the ground of *all* diseases which either directly or indirectly are associated with animal contacts, but the following Table (I) gives an indication of some of them. Many are already familiar. Some however are not so well known, and it is with two of these in which work has been done in the Bristol area that I wish to deal: Canicola Fever, which is contracted from dogs, and toxoplasmosis, which may be contracted from a variety of animals. Before passing on to discuss these in more detail there are three points worth stressing about other infections. Many animals suffer from salmonella infections or are carriers of organisms pathogenic to man, and

* A communication to the Bristol Medico-Chirurgical Society on Wednesday, 14th March, 1951.

TABLE I

Animal Source	Disease	Organism	Frequency	Method of Infection	Symptoms
RAT	(i) Rat bite fever	Bacillus Spirillum	Rare	Bite of rat	Undulant fever jaundice, nephritis, meningitis
	(ii) Leptospirosis	Spirochaete	Not uncommon	Direct (bathing)	Enteritis
	(iii) Salmonella	Bacillus	Not uncommon	Direct or indirect	Enteritis
	(iv) Plague	Bacillus	Common in Asia	Indirect, via flea or tick	Pneumonic, bubonic
	(v) Typhus	Virus	Common in Asia	Indirect, via flea or tick	Typhus (Typhoid-like illness)
MOUSE	(i) Leptospirosis murina (ii) Salmonella	*See* above			
	(iii) Choriomeningitis	Virus	Rare	Indirect	Meningitis
DOG	(i) Tuberculosis (human)	Bacillus	Rare	Direct	Usually phthisical
	(ii) Canicola fever	Leptospirosis	Rare	Direct	Meningitis, Conjunctivitis
	(iii) Hydrophobia, Rabies	Virus	Rare except in Asia	Direct (bite)	Encephalitis, myelitis
	(iv) Toxoplasmosis (*see* Rabbits)	Protozoa	Rare	?	
	(v) Hydatid disease	Worm	Rare except in Wales and Scotland	Eating soiled vegetables	Cysts in various organs, lung, liver, etc.
	(vi) Ringworm	Fungus	Rare	Direct	Skin eruption
BAT	(i) Hydrophobia	Virus	Rare (in Trinidad)	Bite of Vampire Bat	(*See* Dog)
BIRDS	(i) Psittacosis (parrots, love birds)	Virus	Rare	Direct	Pneumonia
	(ii) Toxoplasmosis	Protozoa	Rare	?	(*See* Rabbit)
	(iii) Salmonella (duck, fowl)	Bacillus	Rare	Indirect from eggs	Enteritis

TABLE I

Animal Source	Disease	Organism	Frequency	Method of Infection	Symptoms
HORSE,	(i) Glanders	Bacillus	Very rare	Direct	Respiratory or dermatological
	(ii) Equine encephalitis	Virus	U.S.A. only	Direct: or indirect via insects	Encephalitis
	(iii) Anthrax (also Sheep and Cow)	Bacillus	Not uncommon	Direct or indirect	Pustule and respiratory
COW	(i) Brucellosis	Bacillus	Not uncommon	Direct: or indirect via milk	Undulant fever
	(ii) Bovine Tubercle	Bacillus	Not uncommon	Direct: or indirect via milk	All types (bone and joint)
	(iii) Salmonella	Bacillus	Not uncommon	Direct: or indirect via milk	Enteritis
	(iv) Taenia Saginata	Worm	Rare	Eating infected beef	Tapeworm infestation
	(v) Ringworm	Fungus	Rare	Direct	Skin or scalp infection
	(vi) Anthrax (*See* Horse)				
GOAT	(i) Brucellosis	Bacillus	Common only in Malta	Direct: or indirect via milk	Undulant fever
SHEEP	(i) Louping ill	Virus	Rare	Direct: or indirect via milk	Meningo-encephalitis
	(ii) Anthrax (*See* Horse)				
PIG	(i) Brucellosis (*see* Cow)				
	(ii) Swine Fever	Virus	Switzerland	Direct	Influenza
	(iii) Taenia Solium	Worm	Not uncommon	Eating infected meat	Worm in gut or cystic (epilepsy)
	(iv) Trichiniasis	Worm	Common in epidemics	Eating infected meat	Cysts in muscle etc.; ac. myositis
HARE, RABBIT	(i) Tularaemia	Bacillus	U.S.A. and Russia	Direct: or indirect via tick	Pneumonia, rash, high fever
	(ii) Toxoplasmosis	Protozoa	Rare	?Direct or indirect	Meningo-encephalitis, Eye infection

there is considerable evidence that birds and animals thus infected may either directly or indirectly by contamination of meat in slaughtering, pass the infection on to humans: this accounts for some epidemics of food poisoning. Dogs can suffer from human tuberculosis and infect man: recent work in Sweden traced an infection of a whole family to a dog severely infected with tuberculosis, which in turn had previously contracted the infection from its former master who had died of open phthisis. Last in this category is the work done in America linking the epidemics of human encephalitis with equine encephalitis, in the transmission of which mosquitoes, tics and birds all have a part. Although equine encephalitis is not found in this country it might develop here at any time, so it is wise for us to remember this in a case of encephalitis occurring in a person working with horses.

Canicola Fever

Canicola fever is an acute infective disease cause by the *Leptospira Canicola*, an organism of the same genus as *Leptospira Icterohaemorrhagica* which causes Weil's disease. But whereas in the latter the disease is contracted from rats, the former is a parasite of dogs. When it infects man it causes an influenza-like and meningitic illness, often complicated by eye symptoms. It is most prevalent in Flanders: but from the incidence of some twelve to fifteen cases in Bristol in the last two years, and one at least from the Dursley area, it is obviously quite frequent in the British Isles. This disease is common in dogs, particularly in large towns: about 30 to 50 per cent. of all dogs in towns have been infected, and we have demonstrated that an infected bitch can infect her own puppies. The organism is found in the urine in active cases, and two of our patients were infected by contact with urine, having had to mop up the urine of infected puppies. In one case the infection may have entered through an abrasion. We obtained evidence that dogs in the Shirehampton area were heavily infected, and at least five human cases have come from this area.

The disease usually presents as a meningitis and various neurological complications may take place such as paralysis of the cranial nerves or of the legs. Eye symptoms are common, such as mistiness of vision, iritis, diplopia and optic neuritis. The

diagnosis is made by the appropriate serologicalr eactions. The disease is severe in dogs, and causes death by renal failure. Death is rare in man, but three cases are recorded, all due to uraemia. In dogs and man treatment by penicillin and streptomycin is effective. Most cases occur by direct contact with infected animals. But there is ample proof that (as in Weil's disease) infection may arise from bathing in pools or rivers contaminated by infected dogs. The preponderance of the infection in boys emphasises the greater fastidiousness of the fair sex in the choice of a bathing place. With more care in dealing with dogs' excreta, the prevention of vegetables becoming soiled, and the avoidance of dirty bathing places, this disease need never become a human problem.

Toxoplasmosis

Toxoplasmosis has only recently been recognised in England. In animals the disease has been known since 1908 and has been found in many species. It occurs in rats, rabbits, dogs and squirrels and it is probable that animals infect man, although exactly how this occurs is unknown. The organism is a crescentic protozoon which can invade any tissue but has a predilection for the tissues of the nervous system and eye. The organism multiplies by simple binary fission and is intra-cellular, sometimes forming groups or pseudo-cysts with clusters of organisms which may degenerate to form a necrotised granulomatous mass.

Although the organism was first described by Jankû (1923) most of the credit for investigating toxoplasmosis must be shared by Wolf *et aliis* (1939) and Sabin (1941) in America. Wolf first demonstrated animal infection from human material and the congenital nature of the disease. Sabin has classified the disease into four main groups:

(i) Congenital.
(ii) Acquired disease in children.
(iii) Acquired disease in adults.
(iv) Latent disease with only pathological but no clinical findings.

The congenital cases show symptoms at birth and there may or may not be clinical evidence of the disease in the mother.

Sabin has described a triad of symptoms that he considers important in the clinical diagnosis: hydrocephalus, cerebral calcification and choroido-retinitis. It is obvious that only one of these symptoms may be present and the case may still be one of toxoplasmosis. Callahan (1946) found in St. Louis in 10,000 autopsies five cases of infantile toxoplasmosis which previously had passed unrecognised; this sort of incidence is probably true of other cities. Apart from the clinical recognition of the disease diagnosis rests on the finding of the organism (which is extremely difficult) and on demonstration of antibodies in the patient's serum. The value of the serological and pathological tests in this condition have been fully reviewed by Cathie and Dudgeon (1949). Evidence is now accumulating that many persons show immune bodies to toxoplasmosis, and Sabin (1950) found figures as high as between 10 and 60 per cent. of positive serological tests in " normal " people in certain American cities. Before a diagnosis of toxoplasmosis can be reached it is necessary to weigh the clinical and pathological evidence most carefully.

The Disease in Adults. Sabin (1942) described an acquired form of the disease in older children, stressing the occurrence of acute encephalitis. As early as 1914 Castellani had claimed to have found an organism resembling toxoplasmosis in a Cingalese boy of 14 years, dying of an unknown fever with splenomegaly and anaemia. Pinkerton and Henderson (1941) described an acute illness in adults which resembled typhus fever, in which there was a macular rash, pyrexia, and pulmonary signs; and in whom toxoplasma were demonstrated at the post-mortem. Their cases showed a resemblance to the Rickettsial diseases, and in both cases was a history of a bite by a tick. In one of their patients the disease was successfully transmitted to guinea-pigs. Guimaraes (1943) described a young Brazilian negro who died of an acute meningo-encephalo-myelitis at the age of eighteen. At post-mortem the patient showed extensive inflammatory areas throughout the brain from which toxoplasma were isolated.

Syverton and Slavin (1946) described an illness in a 65-year-old tailor with fever, diarrhoea and joint pains, a macular rash and a persistent eosinophilia of from 25 to 45 per cent. Biopsy material from this case showed it to be a case of toxoplasmosis. This man had had an unexplained rash twenty-five years

previously which may well have been a manifestation of toxoplasma infection. It suggested that the disease might remain latent for many years, and that, as in the cases we propose to record, fluctuations of the rash and suffusion of the eyes may have been an allergic reaction to toxoplasma antigen. Brennan (1950) has suggested that increase in the severity of the rash coincides with the release of new toxoplasma antigen from an internal focus. Binkhorst (1949) records in the mother of one of his cases the occurrence of a rash during her pregnancy which may have been due to toxoplasmosis, and the mother of the case recorded by Wyllie (1950) had a similar rash.

The familial side of this disease has not been adequately studied. The pathological tests have indicated that in affected infants and children the mother has often shown either clinical signs of choroido-retinitis, which were previously undetected, or positive antibody reactions. Zuelzer (1949) has recorded the case of twins who suffered from the disease, and Robson (1950) has studied the case of a father and child who had both had a fever and encephalomyelitis; but the father's case history is somewhat indefinite and his case cannot be definitely regarded as one of toxoplasmosis, although several members of this family had choroido-retinitis and positive serological tests.

In the Bristol area two families with toxoplasmosis have been investigated. The signs and symptoms of the first family are fully outlined in Table II, and this family illustrates very well the adult type of the disease. The second family consisted of a clinically normal, but pathologically positive, mother whose first child is in the Blind School with hydrocephalus, choroido-retinitis, optic atrophy and dementia: her second child died three days after birth of jaundice, probably from toxoplasmosis, and a third child, now three months old, showed positive serological tests at birth. This case is still under investigation.

To return to the first family, which has many unusual features. The picture of these cases can be compared to the behaviour of congenital syphilis, and there is evidence of transmission through three generations. It is impossible to say with certainty whether the disease originated in the mother of the original patient, or his grandmother. This lady has shown one positive antibody test only. The infection has remained present in a sub-acute form in

TABLE II (First Family)

Comparison of three members

	Mrs. C.	*R.C., son*	*Mrs. G., daughter*
Headache and sickness ...	+ since age of 5	++ since age of 8 Very acute recently	++ since age of 14
Rash intermittent)... ...	++	++	++
Suffusion of conjunctiva ...	++	++	++
Pyrexia	−	+	−
Saddle-back Nose ...	+	+	+
Claw Feet	−	+	+
Joint Pains	+	+	+
Cough	+	−	−
Hydrocephalus	−	+	+
Intra-cerebral calcification	−	−	−
Micro-ophthalmos ...	+	−	−
Micro-cornea	+	−	−
Choroido-retinitis ...	+++ macular and peripheral	+ slight peripheral	++ peripheral
Optic atrophy	+++	++	+
Papilloedema	−	++	+

three members of one family, and possibly four, for many years, causing chronic ill health, headaches and the symptoms shown in Table II. It appears that a state of chronic toxoplasma infection can exist in adults where the antibody response is insufficient to deal with the infection. In most persons infected the antibody response is obviously adequate, with the exception of the foetus in utero which is very liable to a severe and fatal infection. As yet we have no knowledge of how man may acquire the infection: but it is becoming increasingly evident that dogs may become infected and their proximity to man makes them a most likely source of human infection. Recently the disease has been transmitted to guinea pigs by vaginal inoculation.

TABLE II (FIRST FAMILY)

members most affected

	Mrs. C.	*R.C., son*	*Mrs. G., daughter*
Opaque nerve fibres ...	−	+	+
Nerve deafness	+++	+	++
Meningo-encephalitis ...	++	++	+
Spasticity of legs	+	+	−
Extensor plantar reflex ...	+	+ bilateral	+ left only
Splenomegaly	+	−	+ temporary only
Wassermann Reaction ...	−	−	−
Blood ...	−	−	−
C.S.F. ...	−	−	−
C.S.F. Pressure	Normal	300 mm.	Normal
Pleocytosis... ...	20–30	80–190	20–30
Protein, per cent.	110–160 mg.	70–95 mg.	20–65 mg.
Eosiniphilia			
Blood: per cent. ...	4–8	10	4
C.S.F.: per cent.	Unknown	5–10	6
Cytoplasm modifying test...	+ at 1/32	+ at 1/128	+ at 1/32
Complement fixation test...	+ at 1/8	Eight times	+ at 1/16
	+ at 1/16	+ at 1/16	
		+ at 1/8	
Toxoplasmin Skin test ...	Not performed	+ 24 hours	+ 24 hours
		+ 48 hours	+ 48 hours

It may well be that certain persons are more susceptible to infection than others, and this would explain the large number of positive antibody tests in apparently healthy persons. The persistence of proliferation of toxoplasma in the eye and central nervous system may be explained by the poor diffusion of serum antibody into these tissues. The family recorded must have had a poor antibody response. As has already been suggested, the continued release of antigen accounts for the rash, uveitis and eosinophilic response seen in the patients.

The case recorded by Brennan (1949) resembles this family most closely in that a man of thirty was found to have the same generalised cutaneous eruption, choroido-retinitis and eosinophilia,

with persistent but fluctuating headaches, with positive antibody tests and positive intra-dermal tests for toxoplasma antigen. This patient also had an intermittent fever, as shown by the patient, R.C. The eosinophilia was 4 per cent. This patient appeared to improve on sulphonamides, in the same way as R.C., but relapsed when they had to be discontinued due to haematuria.

The nerve deafness found in all these case has not been previously recorded in toxoplasmosis, and it is important for aural specialists to bear toxoplasmosis in mind when examining cases of nerve deafness in children and young adults. The choroido-retinitis shown by these cases is much more slight and peripheral than the gross lesions seen in some cases where there is a large area of choroido-retinitis near the macula. The pepper-and-salt appearance of small lesions in these cases was much more marked in Mrs. C. than in her children. Therapy presents a most difficult problem as most drugs, with the possible exception of sulphonamides, have little effect on the organism. Frenkel (1949) has reported some improvement in retinal lesions with graduated doses of toxoplasma antigen, and it might be worthy of trial in the case R.C.: but we fear that many of the changes in these chronic cases are irreversible. The poor results of therapy are strictly in accord with the experience of other workers.

In conclusion, this paper has merely touched on a fascinating problem in epidemiology and public health. Such questions as whether " swayback " is related to disseminated sclerosis, or whether there is an animal vector or reservoir of the poliomyelitic virus, remain unsolved. Nor do I advocate our casting away our animal friends: rather that we remember we can get disease from a sick animal or a sick bird, and we should take the same precautions with them to avoid infection as we would take in a case of a known human infectious disease, particularly where food and excreta are concerned. We should as a profession have more contact and discussion with veterinary surgeons about prevalent animal epidemics in relation to human disease. The two professions can learn a great deal from each other, and in the past this fact has been neglected. It may be of interest that a good observation by a Weston-super-Mare doctor has, since

this paper was read, shown a child with paratyphoid B. had become infected by her pet tortoise.

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THE JOURNAL: A SWAN-SONG

BY

E. WATSON-WILLIAMS, *M.C.*

Reporter to the Bristol Medico-Chirurgical Journal 1922; *Editorial Committee* 1926; *Assistant Editor and Editor of Jubilee Index* 1933; *Editor* 1948; *Sometime Honorary Treasurer of the Society of Medical Editors of Great Britain and Ireland*

Our first editor began the tradition that each occupant of the chair should write something about medical journalism. Provincial medical journals have never been numerous in this country; many have perished young. As we shall see, the 1914–18 war caused several old-established journals to suspend publication, although some (not all) resumed about 1926. Even of these, few overcame the mischances of the last ten years. And apparently, if we exclude annual reports of hospitals, transactions of societies and so forth, ours is the only English provincial medical journal to have survived both World wars. In this at least we may feel a modest pride.

The origin of the *Journal* has been well described by L. M. Griffiths (1900: Assistant Editor 1883, PRESIDENT 1887, HONORARY MEMBER), who had himself contributed very largely to it.

" In 1878 it was decided that some of the good work which the Society had done should find a permanent record in a volume of Transactions. Accordingly a volume was issued which, considering the quality of the matter it contained, was published at the exceedingly modest price of five shillings.

Another four years passed, and some members of the Committee thought the time had arrived for the publication of a second volume of Transactions covering that period. Feeling that such a retrospect was at its best by no means satisfactory, and knowing well the ability of my professional brethren in the district, I had the temerity to propose in Committee that a publication with a more frequent periodicity and having the nature of a journal or magazine, should be attempted. This proposition received the support only of Greig Smith and myself. But when the question introduced by Dr. Beddoe was brought before a general meeting of

the Society it was determined *nemine contradicente* that the effort should be made, and in 1883 the first number of the *Journal* was issued. It was fortunate in its beginning. In the person of Greig Smith it had an Editor who *con amore* placed at its disposal his commanding and masterly genius.

The second number appeared after an interval of six months, but after the first year it became and has remained a quarterly publication. Its professional success has been great. This is partly testified by the fact that its contributors often find abstracts of their work chronicled in various countries of the world, and partly by its large exchange-list; for, with the exception of some few journals of a special nature which exchange only with their own kind, this includes most of the best medical periodical literature extant. An examination of the exchange-list and of the list of subscribers shows that the *Journal* now travels over the whole globe.

My journal career has shown me that in the work of such a publication there is room for the man of small parts. A successful medical journal must have at the head of its affairs an editor with a well-balanced mind, and among its staff and its contributors those who have clinical and literary accomplishments; but, in addition, it must have someone who will look after its business affairs, see that its matter is methodically arranged and in something like right proportions, and that it goes out with every "i" dotted, and with every "f" unbroken. I part from my work (as Assistant Editor) with many regrets, though it occupied more time than perhaps I was justified in giving to it. I have felt all along that the professional life of a district is the richer for a *Journal*, which worthily gives expression to that life."

Here is J. Greig Smith, our first editor (Editor 1883, PRESIDENT 1893), expressing his opinion on "Modern Medical Journalism" (P.A. 1893) in no uncertain manner:

"There are sundry difficulties which meet the best-intentioned editor which he cannot remove, and which he must accept. One of these, I grieve to say, relates to the deficient literary education of the average medical man. For some mysterious reason the art and science of logic is not expected to be known by medical men. It is not known, and their writings show it. The arts of rhetoric and composition also are ignored; even the humdrum rules of grammar are in abeyance. How much of the editor's function is the humble one of correcting a school exercise in grammar and composition the editor only knows. It is certainly much more than it ought to be, and much more than it need be, if those who guard the portals of medical education were to give some heed to the outcry of those who practise their profession and teach it.

This, then, to begin with. We must accept the fact that, with a few, a very few, and sometimes brilliant exceptions, the literature of medicine at the present day is, as pure literature, either indifferent or positively bad. Scarcely a single issue of a journal is without a paper from which a man

educated in ordinary literary methods could not extract abundant examples of errors in logic, in rhetoric, in composition, and even in humble grammar.

Another fact which the editor must accept is the wide-spread ignorance of the history of medicine—recent and remote—which his contributors display. Such ignorance betraying itself on every hand lends colour to the view of certain cynics who hold that it is only by courtesy that our profession is called " learned "; that we are educated by rote, and not by thought; that the evolution of ideas has no interest for us; and that our classics, being dead, may as well be buried. There is ignorance not only of classics, but even of class-books. It would truly seem as if knowledge in our profession began to decay after five years, and was buried and forgotten after ten. However this may be, it is certain that too many writers show a culpable ignorance, or neglect, of the works of others in the fields they seek to cultivate.

The man who earnestly and honestly seeks to keep abreast of real knowledge has to educate himself in a course of training in values whereby he finds out and knows the fool, and the drudge, and the bore and the advertiser; and learns to avoid them. When he has done this, he finds that his labours are not so hard after all. It is, then, a mere turning over of thousands of leaves and a close reading of tens of pages. But it takes a long time, and greatly tries one's patience, to learn whom to avoid in medical literature.

And here our editors might help us. They might select for us. They might silence the chatterer and make the worker talk. The best men and the best workers are silent and retiring; the editor must seek them out, and drag them out, and make them speak. This is not easy matter, as I can from experience testify. It is very hard to get our best men to write."

In the same address Greig Smith spoke of his early elevation to the Presidential Chair as a " reward " for his services as editor. In actual fact this honour has, from our first years, been the traditional due of the *assistant* editor. His post is no sinecure. A mere list of the more important duties would occupy well over a page of this *Journal*: even such a glutton for work as Griffiths had to ask for help.

An Editorial Secretary was accordingly appointed in 1895 to take part of the burden. Curiously, he seems from the first to have been considered more important than the A.E. For example, for the next thirty years *he* presents the annual report. The Editor of course (as now) was appointed by the committee of the Society, not annually but " during pleasure ", a provision necessary to secure continuity. But he was not himself a member of that committee, whereas the E.S. seems to have been elected to it each year. The position of the A.E. is obscured by

the fact that he was for years Honorary Librarian, and as such *ex officio* member of the committee.

In 1908 new rules provide that the Editor (appointed as before) and the E.S., elected annually, are *ex officio* members of the committee. And each year until 1913 the A.E. seems to have been one of the *elected* members. From then until 1923 no accounts of meetings were published, except for 1919, when the same plan held. In 1923–24 the A.E. was apparently elected as " Past President ". And in 1925 new rules abolished the office of Editorial Secretary. With the indifference of Gallio, the *Journal* continued each year to print the name of the E.S., Dr. A. L. Flemming, who discharged the duties until his death in 1939: the annual report, however, was presented by the Editor or the A.E. Perhaps because in 1925 the E.S. was President, and the A.E. became Editor, no one noticed that the *Journal* had lost half its former representation on the committee. While the office lasted the following held it:

B. M. H. Rogers. Editorial Secretary 1895; PRESIDENT 1910.
J. Taylor. Editorial Secretary 1899; PRESIDENT 1906.
J. A. Nixon. Editorial Secretary 1909; PRESIDENT 1922.
J. M. Fortescue-Brickdale. Editorial Secretary 1913.
A. L. Flemming. Editorial Secretary 1921; PRESIDENT 1926.

R. Shingleton Smith (PRESIDENT 1888, Editor 1892, HONORARY MEMBER), who succeeded, did not write about journalism, perhaps feeling that Griffiths had said all that was required. During his long editorship the *Journal* developed considerably. The work of Lister and the discovery of bacteria had ushered in a medical renaissance. 1883 was the beginning of *The Medical Annual* as well as of our *Journal*. In the early years the bulk of our material consisted of papers and clinical records. But from the very start " Notes on Books " and " Medical Extracts ", a review of recent advances, had found a place.

Under Shingleton Smith the proportions changed. Taking volumes X–XIV, we find only a third is given to original articles. " Progress of the Medical Sciences " and Reviews together

occupy more space than this: and in addition there were quite considerable Library Reports, accounts of various Health Resorts, " Scraps ", and reports on " Meetings of Societies " and " Preparations for the Sick ". Under the last head we find not only descriptions of new drugs and appliances, but an occasional hint of some compensations. For example:

" *Preparations for the Sick* "

Volume	XIII	XIV	XV	XVI
Wines . . .	2	3	1	1
Whisky, etc. . .	–	1	2	1

The note of restrained wistfulness compels me to quote the last report in full:

" This brand of Scotch Whisky bears on its label the descriptive epithets " real and rare ", " very old ", and " guaranteed pure ". *Its flavour and the absence of after-effects** seem to justify these appellations." Ah, me!

The great increase in the labour of writing abstracts, reviews and reports made necessary the appointment of an Editorial Committee (1891), the function of which has always been " to assist the Editor ". The Editor, of course, remains solely responsible for the conduct and contents of the *Journal*: he need not seek the advice of the Committee, nor take it when sought—there is never any question of a " vote ". Economic pressure led to the abandonment of " Notes on Preparations for the Sick " in 1921 and of " Progress of the Medical Sciences " in 1924. Mainly for similar reasons we have quite recently had to cease publishing " Reviews of Books ". Perhaps, therefore, there will no longer be the former need for an Editorial Committee.

" Meetings of Societies " made its first appearance in Volume IX (1891), with fairly full reports of the discussions at the meetings of the Bristol Medico-Chirurgical Society. Although our origin, from " Transactions ", implies that the bulk of what appears in the *Journal* had already been presented to the Society, it is not possible before this date to discover how much. An addition was made at this stage to the Society's " Rules ", giving

* Our italics.

the Editor the right to publish what was said at meetings. The last issue of Volume X carried a report from the Plymouth Medical Society, and others soon followed.

" Local Medical Notes " began in 1896 (XIV, i) when " Local Representatives " of the *Journal* were appointed in Bath, Cardiff, Exeter, Gloucester, Newport, Plymouth, Swansea and Torquay. In the same volume we find a record of the first use of X-rays in Bristol, and a centenary notice of Jenner with his portrait.

An " Editorial Note " in 1892 (X, 72) records the change in editorship; a second, the appointments just mentioned. After 1900 (XVIII) Editorials became a regular feature.

P. Watson-Williams (Assistant Editor 1900, Editor 1912, PRESIDENT 1913, HONORARY MEMBER), succeeded Shingleton Smith in 1912. His " History of the Bristol Medico-Chirurgical Journal " (1933) appeared so recently that it would be idle to quote from it here. It contains much information about format, printing and the contents of early numbers.

J. A. Nixon (E.S. 1909, A.E. 1912, PRESIDENT 1922, Editor 1926, HONORARY MEMBER), delivered an address on " Provincial Medical Journals " (1932) before the History of Medicine Section of the British Medical Association at the Centenary Meeting. In this he summarised the objects and advantages of a local medical journal which I quoted in our last issue (p. 98). Both the last two editors had to face the difficulties involved in conducting the *Journal* during a major war. These were the subject of comment at the time and have been briefly compared quite recently (1947).

Another change introduced in 1926 was the appointment of the Treasurer of the Society as *ex-officio* business manager of the *Journal*. Whether this is altogether fair is open to question: the two functions are essentially different, and their combination may well deprive one institution or the other of the services of a member who feels them incompatible.

My own very short tenure of office has been remarkable chiefly for a persistent economic struggle, due to a continuous rise in the cost of paper, typesetting, printing and distribution. No sooner did the increase in subscriptions and in advertisement

income promise us some relief than a further advance in the expenses of production snatched it away. In an endeavour to demonstrate just where and how " the money goes ", we have regularly published the Balance Sheet of the *Journal*, and with it an " analysis " which brings out clearly that the number of words printed is the determining factor in the cost of production. To reduce this we have eliminated Reviews, Lists of Books Received, Obituary notices and most of the examination results: and can hardly go further in this direction. Too often, alas, we have had to refer to the need for economy, the absolute necessity for brevity, the menace of bankruptcy: for without constant care it would be quite easy to squander a whole year's subscription income on a single quarterly issue—we came within a few pounds of this in 1946. I fear I shall be remembered, if at all, as the Editor who was mad about economy!!

As a more positive attack on our economic difficulties, the appointment of Local Correspondents has been revived: a number of colleagues having kindly undertaken to promote our interests in their areas. At the same time we have tried to devote more space to Local Medical Notes and Proceedings of Societies, and to show that our interests are not confined to Bristol. Many of the senior practitioners in the region were approached and in addition some hundreds of former Bristol students. The mere compilation of the list of the latter was an extremely tedious business and the response not encouraging. Further, in addition to reporting the papers contributed to our own and other societies we have endeavoured to introduce matters of general interest, such as the reports on the Bristol Quadruplets and on " Unusual Children ". A series such as this entails quite a lot of work: we hope it was worth it. At least at the moment the note of moderate optimism in the Editor's Report on page 139 appears to be justified.

We have succeeded also in securing an additional amenity for those subscribers to the *Journal*, whether members of the Society or not, who live at a distance from Bristol. By virtue of a recent agreement they are now entitled to borrow books from the University Library by post.

The other factor that has recently dominated our professional life has been the Health Act. We are all rather conscious of the

difficulties inherent in such a fundamental change, particularly on the administrative side: and have been several times reminded of the stringency of materials and of finance which must continue to limit expansion. It is too early to assess how this change will affect medical journalism, although already we can detect an increasing absorption in affairs which tends to make some of our best men reluctant to serve the *Journal*. How interesting now to recall the views expressed by Lord Dawson (1920), Chairman of the Consultative Council, Ministry of Health, when he visited Bristol in 1920 : " A municipalised (Hospital) service was one step forward to the nationalisation of medicine, and that would be a disaster alike to the medical profession and the public."

My own connection with the *Journal* began in 1922 when I was asked to undertake the duties of Reporter of the discussions of our Society: a duty which I continued for some years after joining the Editorial Committee in 1926. The first meeting of that Committee which I attended was number 349 and the last, number 541. After so long a probation it would be scarcely human not to feel disappointment that the tenure of office has been so short. " The labour we delight in physics pain ": but we like the labour to be recognised as well as the delight.

> *αἰνὸν ἄχος τό μοί ἐστιν, ἐπεὶ πάθον ἄλγεα θυμῶι.*
> *ἀλλὰ τα μεν προτετύχθαι ἐάσομεν. οὐδ' ἄρα πως ἦν*
> *ἀσπερχὲς κεχολῶσθαι ἐνὶ φρεσίν. ΙΛ. Π*

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THE DIAGNOSIS AND TREATMENT OF CONGENITAL HEART DISEASE*

BY

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I—DIAGNOSIS AND MEDICAL TREATMENT

BY

JOHN APLEY

Is congenital heart disease rare or common? The question is well worth an answer before turning to the fascinating problems of diagnosis. Until recently it was accepted and taught that among children with organic heart disease only one case in ten was congenital in origin, with the remainder almost exclusively rheumatic. Recent surveys tell a very different story.

Autopsy incidence. News tends to grow less impressive with every mile it travels, and local information is always more interesting than that from a distance. Fortunately, therefore, autopsy figures are available from local sources. With Professor Hewer's permission I have examined the post-mortem records at the Department of Pathology, relating to deaths at the Bristol Royal Infirmary, General Hospital, Children's Hospital and Southmead Hospital, over the last ten years. It must be appreciated that they are concerned not with a general, but with a hospital population, and one, moreover, with an obstetric bias.

In 5,000 autopsies there were congenital anomalies of the heart in 108, roughly a proportion of one in fifty, or 2 per cent.

* Papers read to the Bristol Medico-Chirurgical Society, with X-ray films to illustrate the conditions, on Wednesday, 9th May, 1951.

In this total all live births have been included, but an appalling number of infants died within a short time of birth: one quarter (twenty-seven cases) did not live as long as one month, and more than half (sixty-seven cases) died within the first year. In a small proportion of cases the cardiac malformation was not the cause of death. In a survey of post-mortem evidence from world-wide sources, though apparently none has hitherto been available from this country, Brown (1950) also arrives at an average incidence of 2 per cent.: but he utters the caution that the inclusion of figures from children's hospitals and neo-natal departments tends to give an unduly high incidence.

Cases at a cardiac clinic. To clinicians, however, it is more appealing to contemplate patients than corpses, and here too local information is obtainable. It is natural to use rheumatic heart disease as a yardstick and I have had the welcome opportunity of taking part in an analysis of the attendances at Professor Perry's cardiac clinic for children, where ample material is available. At this clinic, over a period of six years (from 1943 to 1948), cases of congenital heart disease were slightly more than half as frequent as rheumatic heart cases. But when it is considered, as we have already seen, that many infants with congenital heart disease die before they are referred to any clinic, it becomes apparent that the proportions are more nearly equal This bears out the conclusion reached elsewhere (Keith,1951), namely, that if all children from birth to school-leaving age are included the proportions of congenital and rheumatic heart disease are roughly the same.

These figures would appear extraordinary to previous generations of medical men, and one reason for the remarkable change in evaluation is that rheumatic heart disease is becoming distinctly less common than it was; another, which concerns us more closely here, is that congenital malformations of the heart are coming to be much more widely recognised.

Relative incidence of cardiac malformations. Like human figures, statistical ones are often dull and sometimes even repellent. To illustrate again our changing perspective I would, however, like to mention one last group of figures. Students are still taught what, until recently, clinicians believed: that simple pulmonary stenosis is one of the common congenital cardiac

malformations. We now know that it is rare. In 300 consecutive children with congenital heart disease examined personally, many by the courtesy of my colleagues and especially Professor Perry, it was considered to be present in only 3 per cent. In 80 per cent. of all cases a definite diagnosis was made, and of these the six commonest malformations were as follows:

Ventricular septal defect (VSD)	23 per cent.
Auricular septal defect (ASD)	15 ,, ,,
Patent ductus arteriosus (PDA)	15 ,, ,,
Tetralogy of Fallot	12 ,, ,,
Coarctation of the aorta	6 ,, ,,
Transposition of great vessels	4 ,, ,,

It should be noted that these proportions are affected by the addition of cases sent to the Surgical Unit at Bristol specifically for operation; at Bath, where this consideration does not apply, the figures for patent ductus and coarctation are less than half as high.

Diagnosis

Since then we are dealing with a fairly common group of disorders, and one in which cure is sometimes possible, it must be accepted that diagnosis is of undoubted practical importance. The new and elaborate methods of investigation are producing results which are important and far-reaching, but these must eventually be re-stated in the language of clinical medicine. It is our task, as clinicians, to use them by giving to physical signs new values and new emphasis, and indeed this is already being achieved.

In nine patients of every ten with congenital heart disease an exact diagnosis can be made if all the ingenious tools of modern cardiology are employed. By combining the simple methods of clinical assessment, cardioscopy (visualising the heart by X-ray screening) and electrocardiography, an accurate diagnosis can be made in some three cases of every four, and these methods should certainly be used in hospital practice. By incorporating the new facts derived from these methods, clinical diagnosis itself is clearly advancing in precision. It would be over-optimistic to attempt to diagnose the more complicated malformations: instead, surely, the clinician should concentrate on the common

anomalies. In the large majority of all cases a comprehensive diagnosis of congenital heart disease is possible, and in the six common malformations a presumptive diagnosis should be attainable in most instances at the bedside.

Problems of Diagnosis. The surgeon's problems are on a different plane from those of the physician. Where operation is contemplated it appears advisable, in all but the most straightforward cases (*e.g.* ligation of patent ductus arteriosus), to confirm the details of diagnosis and to plan operation with the help of preliminary investigations by some of the more elaborate methods, especially angiocardiography and cardiac catheterisation.

For the clinician's purposes, as compared with the surgeon's the problems are relatively straightforward.

(i) *Has the patient congenital heart disease?* The history can be helpful. It may suggest an inadequate blood supply, either systemic or pulmonary, which results in stunting of growth, poor exercise tolerance, breathlessness and the like. Or, especially in infancy, there may be evidence of cardiac or vascular pressure on thoracic viscera. Examination will be expected to reveal significant abnormalities, to be described shortly, and it is hardly necessary to stress that attention must not be confined to the heart alone.

(ii) *What is the type of malformation?* If there is *no* shunt, as in aortic or pulmonary stenosis or coarctation of the aorta, there may be symptoms and signs of diminished blood supply to the affected part, and possibly evidence of a compensatory collateral circulation. If there *is* a shunt, there may be evidence of deprivation of blood in one side of the circulation (systemic or pulmonary) and excess in the other side. In L-R shunt the most useful evidence is found in the lungs and in the right side of the heart. The classical sign of R-L shunt is cyanosis, but other findings are also helpful. L-R shunt, which includes such common anomalies as VSD, ASD and PDA, occurs more frequently than the reverse. Transposition of the great vessels in infants, and tetrology of Fallot in older patients, are the commonest malformations in which R–L shunt occurs.

(iii) *What is the site of the malformation?* This is determined largely from cardiac examination, mainly by the type of enlargement, alterations in valve sounds, and characteristics of any murmurs. But examination of the arteries and the viscera, especially the lungs, is also important.

The history. The infant may have stridor, bouts of dyspnoea, or evident attacks of pain on feeding. General appetite and development may be surprisingly good; but sometimes the child feeds with difficulty, or fails to thrive even if feeding is satisfactory. A history of repeated "lung infections" or "pulmonary

congestion" is not infrequent. Some children have attacks suggestive of petit mal, in which they go pale and are dazed for a short time (Perry, 1931). The older child may tend to avoid strenuous games, become unduly breathless, or feel unwell after exertion. A few patients complain of what are called " growing pains ", presumably due to deficient blood supply to the legs, with such conditions as coarctation of the aorta or a wide ASD. The child may be permanently or transiently blue, the age of onset of cyanosis varying in different malformations; but it must be born in mind that most patients with congenital heart disease are not cyanosed.

General examination. In most cases, and especially after infancy, routine physical examination provides the earliest clue to the presence of congenital heart disease. The first abnormality detected is usually a cardiac murmur, though in diagnosis it is wiser to leave murmers to the last. The evidence from a murmur is always incomplete and may be misleading.

Growth may be impaired, both in cyanotic and non-cyanotic cases, but *failure to gain weight* is often more pronounced.

Cyanosis should be sought in the warm areas of the body, such as the mucous membrane of the mouth, since blueness of the extremities is often due to extra-cardiac causes. Central cyanosis is nearly always evidence not only of congenital heart disease but, more precisely, of a R-L shunt. It may be accentuated by crying, or by cold. In doubtful cases it should be looked for after exercising the patient. In the same circumstances undue breathlessness should be observed, and may be the only symptom to suggest a deficiency of blood to the lungs.

The chest wall merits careful attention, for chest deformities occur very frequently with congenital heart disease. The two conditions may be merely associated developmentally, but in some patients a characteristic deformity can be seen to develop *pari passu* with alterations in the shape and size of the heart.

In the lungs signs of congestion, if infection is absent, suggest the possibility of a L-R shunt. Moist sounds may be surprisingly sparse or absent with malformations, such as pulmonary stenosis, in which there is deficient blood supply to the lungs, even when pulmonary infection or cardiac failure supervenes.

Examination of the arteries may provide important information.

With one exception, the pulse pressure is normal or small in congenital heart disease: the exception is an extra-cardiac shunt. This is almost always caused by PDA, and the pulse pressure, though often normal, is never small and may be so great as to be " water-hammer " in type. A marked difference in the two radial pulses, due to unequal development, sometimes occurs with other cardiovascular anomalies. The femoral pulses should be carefully palpated in every child who is being examined, since their absence is evidence of coarctation of the aorta, a diagnosis which should also be considered if there is visible arterial pulsation above the manubrium sterni, or if the blood pressure in the arms is found to be high in children or adolescents.

Cardiac Examination

Cardiac enlargement in infancy and early childhood is almost invariably due to congenital heart disease. If the apex beat is displaced downwards and to the left it is due to L ventricle enlargement (as in sub-aortic stenosis, and some cases of VSD or PDA); if outwards and to the left it is due to R ventricle enlargement (as in pulmonary stenosis and some cases of ASD). It is important, however, to detect R ventricle enlargement in those cases, too, such as tetralogy of Fallot, where the total heart size is not increased; when with R sided enlargement there has been corresponding underdevelopment of the L side. In these the apex beat is not likely to be displaced, but the sternum or ribs may be seen to protrude forward. In many cases the feel of the cardiac impulse is more helpful than its position: with R ventricle enlargement the cardiac impulse has a " flicking " or " tapping " character, quite different from the " thrust " or " heave " if the L ventricle is enlarged.

Auscultation of the heart sounds at the pulmonary area has become increasingly important in diagnosis. The second sound (P_2) heard in this region is normally somewhat louder than the first sound during the early years of life, and is split into two elements. We are learning to recognise disturbances in the balance of the two sides of the circulation by their effects on the two elements of P_2. In truncus arteriosus, pulmonary atresia, or other malformations where only a single large vessel is

functioning, P2 is heard as a single and peculiarly sharp, almost " bell-like ", sound. If the pulmonary blood flow is reduced, as in the tetralogy of Fallot, the second element is diminished, and P2 is often soft. Where the pulmonary blood flow is greatly augmented, as in malformations with L-R shunt, the second element of P2 may be accentuated, or the two elements heard widely separated. With great accentuation of P2 the accompanying impulse may be readily palpable.

Thrills and Murmurs. The " classical " murmur of congenital heart disease is loud and harsh, is accompanied by a thrill, is systolic in time and maximal near the sternum or at the base of the heart. It is true that a murmur with these characteristics almost without exception signifies congenital heart disease, but often the murmur is by no means " classical ". With the largest defects there may be a soft murmur or no murmur. A thrill is not always detectable, especially in infancy and early childhood, and is not essential for the diagnosis. A diastolic (pulmonary or mitral) murmur is not uncommon, though nearly always it accompanies one in systole. The site of maximum intensity of a murmur may be difficult to determine, especially in babies; moreover it may vary in different individuals with identical malformations, or at different ages in any one patient.

The site of maximum intensity of the murmur is an approximate guide in locating the malformation. In infants it is usually impossible to define the site with accuracy, but in children and adults careful auscultation is helpful as long as it is borne in mind that in several types of malformation the murmur may be loudest at one and the same site.

Common Malformations

The clinical picture with congenital malformations in infants and children may vary from that in adults, but it is often typical enough to permit a presumptive diagnosis. The commoner findings are briefly as follows:

Ventricular septal defect. The child is usually active and symptom-free, though a large, high defect may lead to congestive failure, especially in infants. The chest is not deformed and the pulses are normal or small. The heart is not enlarged. The second pulmonary sound is clearly split, often with

accentuation of the second element. A loud, harsh, systolic murmur is heard, maximal in the third or fourth left interspace near the sternum. In older children it is usually accompanied by a thrill, the proportion of detected thrills increasing with age.

Auricular septal defect. The patient is thin and small, and the lower sternum protrudes, though in milder cases there are no such effects and no limitation of activity. The pulse pressure is small. The apex beat may be displaced outwards, and pulsation over the pulmonary area may be seen and felt. The cardiac impulse is " tapping " to the touch. The second pulmonary sound is widely split and often accentuated. A murmur, surprisingly often of only moderate intensity, is heard between the first and third left interspaces, close to the sternum. A thrill is unusual in early life, but becomes commoner later. A diastolic murmur may also be heard.

Patent Ductus Arteriosus. As a rule, the child is healthy and active, though in a few cases there is breathlessness on exertion or stunting of growth. The pulse pressure may be noteworthy in being wide. The cardiac impulse tends to be diffuse and heaving, and pulsation over the pulmonary area visible and palpable. The second pulmonary sound is loud and split. A harsh, continuous murmur is heard, rising in intensity during systole and fading in diastole. The murmur is usually widely audible but maximal over the pulmonary area, though it may, in a few cases and for some unknown reason, be heard only close to the sternum or under the clavicle. A thrill, either continuous or systolic, is almost always palpable if the typical murmur is present.

Coarctation of the aorta. A number of patients with this malformation, nearly always undiagnosed, die in infancy; those who survive are usually robust and active unless some additional anomaly is present. The diagnosis may first be entertained on hearing a systolic murmur over the precordium or at the back, on finding the blood-pressure to be raised, or on observing arterial pulsation in the neck; it is unusual in young children to see arterial pulsation in the chest wall, though it may be felt, especially if the patient leans forward with the arms hanging down. Coarctation of the aorta is confirmed by the absence of pulsation in the femoral arteries, and if this examination is

regularly practised a number of cases will be found by this sign alone at an early age.

Tetralogy of Fallot. The child has usually been cyanosed from birth or soon after, but always within the first two years of life. He is nearly always thin, and usually under the average height for age. The pulses are normal or small. The sternum or the ribs to its left may protrude forward, though the heart is not clinically enlarged. The cardiac impulse is localised and tapping in character. The pulmonary second sound tends to be soft. Usually, a harsh systolic murmur is heard, which is maximal at the second or third left interspace, and often, especially in older children, accompanied by a thrill.

Transposition of the Great Vessels. The infant is cyanosed, usually from birth, but occasionally not for some weeks. There is failure to thrive, and often difficulty with feeding. There may be dyspnoea and inspiratory recession of the chest wall. In the lungs moist sounds are often heard. The heart is enlarged, and the second pulmonary sound accentuated. Murmurs are variable and may be completely absent, though usually a blowing systolic murmur is heard along the left sternal border.

Medical Treatment

When we come to consider the enthralling subject of surgery in the treatment of cardiac malformations we do well to recall Osler's saying, that: "We stand too close to events to realise their tremendous significance." But obviously the physician, too, has an essential part to play in the management and treatment of the afflicted child.

It is important not to make a "cardiac invalid" of the patient. More often than not, as regards activities, the child adjusts himself to his disability better than the over-anxious doctor or parent does. In general, symptoms are a more reliable guide than are signs, though strenuous games should be barred if the heart is enlarged. A murmur, however loud and threatening it may sound, is not a valid reason for enforcing inactivity. The child should have hobbies and should be educated towards a sedentary occupation if there is a chance of his surviving into adult life.

Contrary to some older beliefs, cardiac failure in congenital

heart disease is frequently recoverable, and should be treated with digitalis and the like. Paroxysmal dyspnoea is effectively treated with morphia. In the cyanosed child fluid should not be restricted, even for operations, for fear of inducing venous thrmoboses. Lastly, if the child has to have teeth or tonsils extracted, the physician may prevent the onset of bacterial endocarditis by prophylaxis with penicillin.

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II—SURGICAL TREATMENT

BY

R. MILNES WALKER

The surgical treatment of disorders of the heart has been one of the startling and sensational events in medical practice of the century. Yet in actual fact the story only goes back twelve years: for it was in 1939 that Gross in Boston successfully ligatured a patent ductus arteriosus, an operation which proved to be the advent of great things to come. It is not my intention here to go into the surgical details of the operations which are nowadays performed for congenital abnormalities of the heart and great vessels, but to say a word about the indication for operation and to give you some idea of the immediate results which are to be expected. As no patient has yet had the opportunity of surviving an operation for patent ductus arteriosus more than twelve years, or for coarctation of the aorta or cyanotic heart disease more than six years, any statement about long-term results would be only a surmise. I think it is safe to say there is every indication that, in the case of patent ductus or coarctation of the

aorta, the patient who survives a successful operation has the prospect of a normal span of life, provided complications had not already set in before the operation was undertaken. And this brings me to a final word in general and that is that there is an optimum time in life for dealing with these conditions and this may be before the patient has any symptoms: for the complications creep in insidiously and many of them may soon reach a state which is irreversible and may thus render the operation more hazardous and the ultimate prognosis more serious.

Patent ductus arteriosus. There are two operations employed for this condition, either ligature of the ductus or else its actual division with suture of the two openings remaining in the aorta and pulmonary artery. In this country the application of double ligatures, one at each end of the ductus, is usually employed as being the safer operation and giving satisfactory results with only a very slight risk of recanalisation. Many American centres adopt the more radical procedure of division, but this requires higher technical skill and it is doubtful if the results justify the additional dissection which is necessary. Crafoord in Stockholm adopts an intermediate course, ligaturing most of them but dividing the short wide ones.

The best age for operation is about five or six years, and any evidence of commencing cardiac dilatation renders operation an urgent matter. It is my own view that in the absence of other cardiac abnormalities all cases should be operated on about this age whether there are symptoms or not, for it has been shown (Keys and Shapiro, 1943) that people who are alive at the age of seventeen with a patent ductus have a life expectancy which is only half that of the general population. The operation, if carried out in the absence of complications, is safe and in itself causes no disability. It is one of the most satisfying operations in surgery for it is one of the few which convert an abnormal condition into one which is completely normal and physiological.

My own series comprises twenty-four cases with no mortality; in only one case was there a serious complication, for I accidentally tore a hole in the pulmonary artery and to close this necessitated division of the ductus; unfortunately I included the recurrent laryngeal nerve in a clamp and the resultant paralysis of the vocal cord made it necessary to perform a tracheotomy

which had to be retained for about a week, but the child eventually made a complete recovery.

There is an interesting point about another patient; she is the oldest patient in the group, twenty-two years, but the point is that her mother had died as a result of a patent ductus which became infected during the puerperium after this patient was born, and we managed to obtain a full account of the autopsy performed at the London Chest Hospital over twenty years ago.

Coarctation of the aorta. As with a patent ductus, many patients with this abnormality live to adult life without great disability but the expectation of life is much reduced and complications may occur without warning. From the surgeon's point of view the main anxiety is that atheroma develops in the wall of the aorta at an unusually early age and thus renders it friable and sutures in it will not hold. This is therefore an argument for early operation; one of our patients, aged seven, already had early atheromatous changes in the wall of the arch of the aorta, and most surgeons are agreed that the hazards of the operation make it unjustifiable after the age of thirty.

The present operation was devised by Crafoord in Stockholm, and first reported in 1945. His operation consists of excision of the stricture and performing an end-to-end anastomosis of the aorta. We have modified this slightly in that I do not excise the whole circumference of the stricture but take out a crescent of about two-thirds of the circumference and leave a bridge of aorta on the inner side so that the whole strain of maintaining the ends in apposition does not fall on the sutures.

Reported series of cases are as follows:

	Cases explored	Coarctation excised	Deaths	Mortality per cent	Later Complications
Crafoord (1951)	85	75	6	8	4 aneurysms
Gross (1950)	100	91	11	12	1 fatality from aneurysm

In six of his patients Gross inserted a graft of human aorta to fill a gap left after the excision and these patients have survived from three months to one-and-a-half years satisfactorily.

Our own cases are listed below:

	Age	Sex	Systolic B.P.	Complications before operation	Operation result
1. M.E.	12	M	160		Well 3½ years
2. I.D.	12	M	160	Intracranial haemorrhage	Well 3 years B.P. 110
3. B.C.	7	M	140		Died—acute tracheo-bronchitis
4. P.S.	8	M	180		Well 6 months B.P. 120
5. M.P.	23	F	220		Well 4 months B.P. 140
6. S.P.	11	F	150	Rheumatic mitral stenosis	Died—rupture of aorta 5th day
7. W.L.	10	M	190		Satisfactory progress 2 weeks B.P. 155

It is my view that operation should be carried out on all patients under twenty who have symptoms or who have a rising blood pressure, unless there is some complication which will make the operation particularly dangerous; it should preferably be undertaken before the age of ten.

Cyanotic heart disease. The credit for instituting surgical means by which additional blood may be brought to the lungs for oxygenation in congenital heart disease goes to Helen Tausig and Alfred Blalock of Johns Hopkins Hospital in Baltimore, and as with coarctation their first results were published in 1945. They felt that if some of the blood in the systemic circulation

could be shunted back into the pulmonary circuit this result would be achieved: and the method adopted was to anastomose one of the subclavian arteries to the corresponding pulmonary artery. Potts of Chicago attained the same result by a direct side to side anastomosis between the descending part of the arch of the aorta and one of the pulmonary arteries, this forming in effect a patent ductus arteriosus. These operations do not, of course, overcome any of the abnormalities existing in the heart and in fact add one more, but the results fully justified the procedure. It is still too early to assess the long-term results, but in Fallot's Tetralogy the change in the colour and activity of the patient is in the majority of cases dramatic.

Cases of pure pulmonary stenosis do not respond so well to a Blalock or Potts operation and for them Brock has devised a direct attack on the site of the obstruction, approaching it through the right ventricle and, in the valvular type, cutting the diaphragm with a knife to make a bicuspid valve, or in the infundibular type punching out part of the obstruction. The results have been most encouraging, and here the actual lesion is being attacked, obviously an improvement on the bypass operation which in fact adds a second abnormality. Brock has carried out in America valvulotomies on some patients who had previously been submitted to a Blalock operation without benefit and after the valvulotomy the Blalock anastomosis has been closed. He is also applying valvulotomy in cases of Fallot's Tetralogy with encouraging results, but how they will compare in the long run remains to be seen. These considerations will convince you how important is an accurate diagnosis before operation.

The following are samples of results which have been reported:

		Operation	*Cases*	*Deaths*	
Brock	Pulmonary stenosis	Valvulotomy	28	8	4 before heart was touched
	Fallot's Tetralogy (only 1 death in last 29)	Valvulotomy	29	2	
	Fallot's Tetralogy	Infundibular resection	24	7	
Sellors (1950)	Cyanotic heart disease	Blalock operation	93	9	

My own experience of cases of cyanotic heart disease is limited and insufficient to provide any conclusions; the first cases were extremely ill and we had some disappointing results. It is now clear that the surgeon must have at his command the experience and the instruments to direct his attack to the lesion in the pulmonary artery or infundibulum or to make a vascular anastomosis outside the heart. Each case must be judged individually and the correct procedure may not be determined until the pericardium is open and the heart can be inspected and palpated. The view is generally accepted that the risk of any of these operations greatly increases as the years pass, and as with the other abnormalities which I have discussed, operation should be undertaken during the second half of the first decade of life.

Experimental work on septal defects. There has been a good deal of enquiry into surgical methods of closing defects in the cardiac septa, but as they have not yet reached the stage of regular clinical practice I will not say anything further about them now.

The number of cases of cyanotic heart disease which will benefit by surgical treatment is not very great and they should be concentrated in a limited number of centres. Recent developments of intracardiac surgery in the treatment of acquired mitral stenosis suggest that this is in fact a matter of much greater magnitude as regards the number of patients to be treated, and that during the next five years it will materially improve their prognosis quite as dramatically as the outlook has changed for those with congenital cardiac defects during the past decade.

REFERENCES

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Gross, R. E. (1950).—*Circulation*, 1, 41.
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Sellors, T. H. and Belcher, J. R. (1950).—*Lancet*, 1950, ii, 887.

Editorial

By the time these pages reach our readers the direction of the *Journal* will have passed into other hands. The Committee of the Society has on this occasion gone back to the principles that directed the first appointment. Mr. H. K. Lucas is a newcomer to Bristol, to the Society, and to clinical journalism. His ideas and ideals have not had time to lose their freshness during a long apprenticeship. We feel sure that all our readers will join in congratulating him on his appointment and themselves on securing his services.

Meetings of Societies

Bristol Medico-Chirurgical Society

Programme for 1951–52

10th October. Annual General Meeting : President's Address.

14th November. Clinical Meeting.

12th December. **"Hiatus Hernia"**, Mr. Ronald Belsey.

Editor's Report

As the balance sheet shows, there has been a slight increase in the cost of production this year, and a slight fall in receipts from advertisements. On the other hand, we have received some arrears of subscriptions from non-members, as well as an increase in subscriptions for the year, and the financial position appears reasonably sound. As a matter of interest we may compare the cost of production of four issues in various years:

1898	£207
1914	£144
1919	£248
1947	£287
1951	£603

But it is clear that we cannot relax any of the restrictions recently adopted. Indeed we have had this year to decide that we can no longer publish obituary notices: we are becoming an older journal, so must expect an

BRISTOL MEDICO-CHIRURGICAL SOCIETY

INCOME AND EXPENDITURE ACCOUNT for eleven months ENDED 31st AUGUST, 1951

EXPENDITURE	£	s.	d.
To Subscriptions to *Medico-Chirurgical Journal*	316	0	0
,, Grant to Medical Library	25	0	0
,, Lewis's Medical and Scientific Library	7	11	8
,, Librarian, Honorarium	15	0	0
,, Treasurer's Secretary	10	0	0
,, Transport of Patients	10	18	9
,, Expenses *re* Meetings	7	7	0
,, Audit Fee	10	10	0
, Printing and Stationery	31	5	11
,, Catering	71	17	6
,, Postages and Sundries	18	6	11
,, Wreath (Professor Nixon)	5	0	0
	£528	17	9
,, Surplus for 1951	93	17	6
	£622	15	3

INCOME	£	s.	d.
By Members' Subscriptions (395 at £1 11s. 6d.)	622	2	6
,, Interest on Bank Deposit Account	12	9	
	£622	15	3

BALANCE SHEET AS AT 31st AUGUST, 1951

LIABILITIES	£	s.	d.	£	s.	d.
Sundry Creditors				101	1	8
Cash due to the *Journal*				153	4	9
Accumulated Fund as at 30th September, 1950	258	15	4			
Deduct: Outstanding subscriptions written off	78	13	6			
	180	1	10			
Add: Surplus for 1951	93	17	6			
Accumulated Fund, August, 1951				273	19	4
				£528	5	9

ASSETS	£	s.	d.	£	s.	d.
Cash at Bank:						
Current Account	270	9	11			
Deposit Account	174	8	5			
				444	18	4
Petty Cash in hands of Librarian				3	5	
Arrears of Subscriptions				83	4	0
				£528	5	9

13 Meadow Street, Weston-super-Mare,
August 23rd, 1951.

Audited and found correct.
C. J. RYLANDS & CO., *Chartered Accountants*

INCOME AND EXPENDITURE ACCOUNT for eleven months ENDED 31st, AUGUST 1951

EXPENDITURE

	£ s. d.	£ s. d.
Arrowsmith, Printing:		
January, 1951	165 4 4	
April	164 5 4	
July	133 6 5	
October (estimate)	165 0 0	
	627 16 1	
October, 1950:		
Estimate ... £150 0 0		
Cost 125 10 8		
Deduct	24 9 4	
	603 6 9	
Wrappers, etc.	12 18 4	
		616 5 1
Postages and Sundries		7 4 3
Audit Fee		4 4 0
		627 13 4
Surplus for 1951		8 17 10
		£636 11 2

INCOME

	£ s. d.	£ s. d.
Subscriptions:		
395 Members of the Society at 16s.*		316 0 0
45 Non-Members		36 0 0
Arrears (non-members)		24 2 6
Miscellaneous Sales, 1945–1950		34 8 6
Advertisements:	£ s. d.	
January, 1951	56 17 10	
April	56 12 4	
July	57 16 0	
October (estimate)	50 0 0	
October, 1950 54 14 0		
Less estimate 50 0 0		
	4 14 0	
		226 0 2
		£636 11 2

BALANCE SHEET AS AT 31st AUGUST, 1951

LIABILITIES

	£ s. d.	£ s. d.
Arrowsmith, for Printing:		
Due September, 1950 ...	494 6 10	
For 1951	616 5 1	
	1,110 11 11	
Advertisements £226 0 2		
Miscellaneous Sales 34 8 6		
Cash Paid ... 659 12 10		
Due August, 1951	920 1 6	
		190 10 5
Auditors		4 4 0
		194 14 5
Surplus for 1951	8 17 10	
Surplus September, 1950 ...	162 14 3	
Surplus August, 1951		171 12 1
		£366 6 6

ASSETS

	£ s. d.	£ s. d.
Cash at Bank		213 1 9
Cash due from Bristol Med.-Chirurgical Society* (included in sum of £316 0 0 above)		153 4 9

Audited and found correct.
C. J. RYLANDS & CO., *Chartered Accountants.*
13 Meadow Street, Weston-super-Mare,
August 23rd, 1951.

£366 6 6

* Although only 395 subscriptions have been received, the *Journal* was sent to 404 members in 1951. Thus there is also due to the *Journal* for 1951 the sum of £7 4s. 0d., added to £7 4s. 0d. for 1950 (*Jl.* LXVIII, p. 29), a total of £14 8s. 0d.

increase in the number of former subscribers for whom such notices would be required—and a notice restricted to a mere few lines is obviously unsatisfactory. We seem now to have reached the irreducible minimum and must hope that no further sacrifice will be needed. We have decided that it should be possible to afford to print the " List of Subscribers " every third year. By omitting degrees and addresses the cost is about halved : this year it amounted to £20.

The number of journals received in exchange for our own is ninety-three, priced at £120. This is a contribution from subscribers to the *Journal* to the University Medical Library.

Plymouth Medical Society

Programme 1951–52

3rd October. Annual General Meeting.

17th October. Dance.

19th October. **" The Medical Witness in Crimes of Violence "**, Dr. F. E. Camps.

16th November. **" Intestinal Obstruction "**, Professor Aird.

January, 1952. **" Peripheral Vascular Disease"**, Mr. Boyd.

Torquay and District Medical Society

President: Dr. Colin MacVicker. *Hon Secretary:* Dr. R. A. Lattey, Ambrook, Rousdown Road, Torquay. *Hon. Treasurer:* Dr. J. R. Imrie.

Calendar, 1951–52

(Meetings Torbay Hospital 8.30 p.m.: unless otherwise stated)

1951, 4th October. 4.30 p.m. **" Industrial Medicine "**, Dr. W. J. Lloyd. 7.30 p.m. Dinner and Dance at the Victoria Hotel, Torquay.

25th October. **" Cortisone and ACTH "**, Prof. G. R. Cameron.

8th November. Clinical Discussion.

22nd November. **" Research in Medicine."**

13th December. **" The Treatment of the Elderly "**, Dr. Marjory Warren.

1952, 10th January. 3 p.m. Clinical Meeting with Branch of the B.M.A.

24th January. **" Modern Aspects of Skin Conditions "**, Dr. John Franklin.

14th February. Clinical Discussion.

28th February. **" Difficult Behaviour in Children "**, Dr. R. F. Barbour.

13th March. 4.30 p.m. Newton Abbot Hospital: Clinical Meeting.

27th March. **"Teaching Medicine."**

24th April. **"Crime Reconstruction"**, Dr. Keith Simpson.

12th June. 3 p.m. Summer Meeting: Royal Naval College, Dartmouth.

South Wales Society of Anaesthetists

A meeting of this Society was held at the Royal Gwent Hospital on Saturday, 30th June, 1951. Demonstrations were given in the morning and in the afternoon. Dr. Musgrove (Cardiff) spoke on **"Hypotension"** and Dr. Rendall Baker (Cardiff) on **"Anaesthesia in Sweden".**

Welsh Paediatric Club

A meeting of this Club was held at the Royal Gwent Hospital, Newport, on 28th February, 1951: the President, Professor A. G. Watkins, in the chair. The first part of the afternoon was devoted to a clinical meeting, various members demonstrating in all some twenty rare and interesting conditions. This meeting was followed by a discussion and then a film demonstration of Rammstedt's operation by Mr. J. T. Rice-Edwards.

West Somerset Medical Club

President: J. F. Coates. *Treasurer:* J. M. Thomson. *Secretary:* C. E. M. Ware.

Meetings first Thursday in each month.

October. **"Animal Diseases Communicable to Man"**, Professor Blakemore.

British Medical Association

Newport Division

During the past year the following programme has been carried out.

October, 1950.—Lecture Demonstration on **"Hypnotism"** by Dr. George King.

November, 1950.—Discussion on **"Peptic Ulceration"** by Mr. Scanlon, Dr. Grahame Jones, Mr. Rice-Edwards and Dr. Wade Thomas.

January, 1951.—Discussion on **"Medicine and the Welfare State".**

February, 1951.—A joint clinical meeting with the South Wales Branch at the new Plastic Unit, Chepstow. Demonstrations by Mr. Emlyn Lewis.

March, 1951.—Annual B.M.A. Lecture **"Legal Hazards in Medical Practice"** by Dr. Forbes, Secretary of Medical Defence Union.

May, 1951.—**"The Extremes of Life."** Joint address given by Dr. T. A. Brand and Dr. Morag Insley.

Local Medical Notes

Honour

O.B.E.—Dr. Elizabeth Casson.

Appointments

Jane Philp, M.B.—Assistant Anaesthetist, Warrington.

G. H. Wattley, M.D., M.R.C.P., D.T.M. & H.—M.O., Grade A, Trinidad.

Bristol : N. J. Brown, M.R.C.P.—Consultant Pathologist.
T. J. Butler, F.R.C.S.—Consultant Surgeon.
K. H. Gaskell, D.M.R.D.—Consultant Radiologist
D. M. Milne, F.R.C.S.—Consultant Thoracic Surgeon.
E. C. Turton, M.B., M.Sc., D.P.M.—Consultant Physician in Psychiatry.
Pamela Westhead, M.B., B.Ch., D.A., Consultant Anaesthetist.

B. A. Jenkins, M.D., M.R.C.P., Consultant Physician, West Cornwall.

H. R. Jolly, M.D., M.R.C.P., D.C.H., Consultant Paediatrician, Plymouth.

A. E. Jowett, O.B.E., M.B., F.R.C.S., Consultant Orthopaedic Surgeon, South Somerset.

J. E. Tees, M.R.C.S., D.A.—Anaesthetist, North Gloucestershire.

R. B. H. Tierney, M.B., Consultant Pathologist, Exeter.

H. R. E. Wallis, M.R.C.P., D.C.H.—Consultant Paediatrician, Bath.

A. E. Wilson, D.O.M.S.—Consultant Ophthalmologist, South Somerset.

University of Bristol

Prizes

Edgeworth.—Miss M. E. Moore.

Mackie.—W. G. Benson.

Western Dental Company's.—J. E. Bowerman.

Phillips.—J. P. Fletcher.

Crosby Leonard.—Miss M. E. Barton.

Tibbitts.—P. Maton Terry.

Degrees and Diplomas

University of Bristol.

M.D.—M. M. Lewis.

M.B., Ch.B. (*with Second Class Honours*).—D. R. Coles (with Distinction in Public Health and in Child Health), June K. Lloyd (with Distinction in Public Health and in Surgery).

M.B., Ch.B.—Margaret M. Ashton, W. G. Benson (with Distinction in Public Health), A. C. Brown, C. T. Brown, Ruth E. Coles, T. E. Dada, C. E. Dickson, Dorothy R. Douglas, M. S. Dunnill (with Distinction in Surgery), M. A Feldman, D. H. Cox, B. W. Hill, Doreen E. D. Hillier, J. A. Jefferies, S. J. Lloyd, Ruth M. Martin, S. J. Palmer, M. E. Parry, D. S. Paton, Margaret R. Salisbury, J. H. Scudamore, M. N. Simmonds, R. E. D. Simpson, R. A. Stride, A. M. Sweeting, Denorah Vardy, P. G. J. Wilcock.

D.P.M.—O. Lyth, W. D. C. Thomas.

L.D.S.—M. F. Flint, Ruth Mulhern.

M.B., Ch.B.—*Second Examination.*—Pamela M. Atkinson, M. T. Charleston, J. F. Daintree, D. J. C. Felton, D. B. Fowler, J. K. Howell, J. S. Hughes-Games, Jean D. Matthews, J. M. V. Packer, Ruth D. Penn, P. J. Roylance, R. M. Stone, D. H. Waldron, G. C. B. Winter.

Royal College of Surgeons.—F.R.C.S.: P. A. James, M.B., Ch.B.

Royal Colleges of Physicians and Surgeons (Conjoint Board).—M.R.C.S., L.R.C.P.: Margaret M. Ashton, T. E. Dada, D. H. Fox, B. W. Hill.

BRISTOL MEDICO-CHIRURGICAL JOURNAL

A Journal of the Medical Sciences for the

WEST OF ENGLAND & SOUTH WALES

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2. Original articles are invited, provided they have not been published elsewhere. For the present we are, unfortunately, compelled to fix a limit not exceeding two thousand words.

3. Notes of forthcoming events, meetings held, degrees conferred, staff appointments, and other local news are welcomed.

4. Illustrations should always be supplied ready for reproduction. All re-touchings or other operations required will be charged to the author. Line drawings should be drawn in Indian ink. We regret that we must ask authors to pay for making blocks, if this is necessary.

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